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* PROBLEM OF DEAFNESS †

with special reference to Otosclerosis and the latest treatment — Rosen's mobilisation of the Stapes.

by

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In a vast country like ours with teaming millions, whose living conditions are extremely unsatisfactory, there are innumerable health problems. There is the problem of preventive and curable blindness. There is the malaria and tuberculosis problem. There is the world-wide cancer problem and so many other problems. But if I say there is also another important problem—the problem of deafness—one would stop and ask what this means for nobody has thought deafness as a health problem.

But if we know the enormous incidence of varying degrees of deafness—if we know the miserable psychologic depression those afflicted by this ailment suffer from, if we are able to appreciate the serious social and financial disadvantages the hard of hearing persons have to undergo, we would certainly pause and say "Yes, it is a real problem".

In the United States alone according to one statistics, there are over 10 million persons afflicted by loss of hearing to various degrees and over a million people use hearing aids. In our country with poor living conditions one can imagine the number of such cases.

The deaf child is a problem for the parents who do not know what to do with him or her. If it is a young school-going child he becomes a problem for the teacher at school who dubs him as a dull boy though he might be as intelligent as any other and there are enough of his classmates to enjoy at his expense. He begins to shun society—begins to lead a lonely life—avoids company and acquires unpleasant habits to attract attention. Psychologically, he develops misbehaviour and unsocial habits. As he or she grows up, the problem of marriage looms large in the minds of the worried parents, who get disappointed, disgusted and highly worried at each proposal breaking off even though they may be very rich.

If, by any chance, he gets through his college career, his difficulties begin anew. He might pass high, but at each stage his future looks dim because his attempts to get footing in the army, in the navy or air a

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force, in the railway or the administrative services, end in failure because the report of the medical examination for physical fitness shows that he is ‘hard of hearing’ and so the application is rejected.

If by any chance he happens to get a position, in any Business Firm or industrial concern, where the strict barrier of defective hearing is slightly overlooked by influence or otherwise, he finds it hard to get going with his superiors and day in and day out, he lives in constant fear of being asked to leave the job because of the defect in hearing. Should he succeed in keeping the job for some years, he finds that many of his juniors with lesser qualifications rise to the top over him but his promotion is stopped and he is where he was when he joined duty.

With this miserable situation, he develops a psychologic change in his facial expressions and social outlook and he goes from doctor to doctor for finding out if there is any ray of hope for his disability. With a sad face, a dejected outlook, a depressed demeanour, he asks the doctor, “Sir, can you do something for me? I am a lost man to society. I have come to you as my last hope. If nothing can be done for me, there is no use of my living in this world”.

There is much difference between the mental factor and psychological changes of the “hard of hearing” and the defective vision cases. At least in many cases of defective vision, there is something that we can do. The refractive errors are easily corrected and surgery has advanced far in eye diseases. To replace a damaged cornea, in some cases of corneal scars, to re-attach a detached Retina, to extract a blindening cataract and so on, are rewards of modern eye surgery. But in the case of the ear, the progress to alleviate the sufferings of the “hard of hearing” has been slow and disappointing because of the peculiar anatomy and pathology of the area.

It is therefore of paramount importance and of great social necessity that the preventive side of deafness should be given great importance in the curriculum of medical studies and educating the public because it is much easier to prevent many forms of deafness than to cure most. Unfortunately the dearth of specialists and the paucity of trained personnel and the light-hearted way things are thought of and done, is in the way of real progress.

Deafness can be acute or chronic; may be mild or advanced-may be partial or complete-may be unilateral or bilaterial-may come on suddenly or gradually-may be temporary or permanent. Here is a wide field for great research-a problem of national and international importance where the public and the State, where the doctor and his patient, where the pediastrician and the physician, the E. N. T. specialist and the Health Services the school teacher and the parent, the Industrial management and the labourer, all have their important role to play to prevent the incidence of deafness.

It is a common question to ask, “Can deafness be cured?”. It is certainly difficult to answer this question. The answer depends on the type of case that presents itself and its duration. It is difficult to answer the question, “Can blindness be cured?”. So is it in deafness.

It is impossible in a short article or talk on the subject to detail all aspects of the cause of deafness most of which a general practitioner is well aware. Research workers have found that over 50% of the cases of deafness in adult life are due to tympanic lesions, resulting from obstruction of the eustachian tubes in childhood. Here the most important role is played by the adenoids. This aspect should be borne by the family physician and should never be forgotten in any work of prevention of deafness.

For the presence of good sight, the cornea, the aqueous humour, the pupil, the iris, the celiary body, the lense and its capsule, the vitreous, the choroid, the retina, the optic nerve and the cerebral cortex and many other factors should be normal. Derangement of any of these will affect the accuity of vision.

So also for good hearing one requires a well shaped clean external meatus with the bony and cartilagenous parts in tact and healthy. The drum membrane with its contour, colour and cone of light normal, the ossicular chain in tact and unaffected with any lesion, the oval and round windows, the semi-circular canals and the intricate inner ear with the endo and perilymph in perfect condition, the auditory nerve function and the cereberal cortex in good order.

If any or many of these component parts are affected, certainly to that extent the accuity of hearing will be lessened. With an atresia of the external canal, with a perforated drum membrane with chronic otitis media, with distruction or dislocation of the ossicular chain and with the auditory nerve lesion, one does not expect to give an answer "yes" to the question "Can deafness be cured?"

For any success in the management of cases of "hard of hearing," one should have a correct personal and family history taken. Personal habits, prolonged use of drugs like quinine, salicylates, streptomycin, alchohol, narcotics, tobacco etc. should be found out. History of frequent attacks of cold, presence of pyorrhae alveolaris, deviation of nasal septum, chronic recurrent attacks of tonsillitis, presence of sinusitis, adenoid growth etc. should be looked for. Incidence of deafness in the family should be elicited. Examination of the urine for albumin and sugar, blood pressure recording, examination of blood for V. D. R. L. and other tests should be done. History of exanthematous fevers, cardiovascular diseases, endocrine disfunction, endo and exogenous toxic states, gastrointestinal disorders, metabolic derangements have all their share of mischief to be looked for.

It is in the experience of many doctors that quite a number of patients come to the clinic with tragic tales of irreversible deafness due to indiscriminate use of modern antibiotics like streptomycin. Whenever one has the necessity to use this drug one should not fail to give the warning to the patient to immediately report if and when he feels discomfort in the hearing apparatus during the course of the treatment.

For cases with any type of deafness, doctors should avoid using arsenicals, solicylatis, etc. In our prescriptions of drugs, it is necessary to think a while if certain damage will be done to certain organs by certain drugs, and a careful personal history of the case is essential. In the management of the "hard of hearing," every point of importance has to be noted, physical defects have to be corrected and focal sepsis should be eliminated.

Otosclerosis: There is one form of deafness about which so much of publicity was recently done and so much of hope given to the disabled persons. I like to mention a few words on this what is called, Otosclerosis, because it is by far very common form of deafness specially in the white race. Research workers have stated that one in 8 white women and one in 15 white men suffer from this form of pregressive deafness. In the United States over a million people seem to suffer from this form of ear defect. It is a big figure. So is it in the continent of Europe. The brown and the black seem to suffer to a lesser extent. The Africans the least. We have no reliable statistics in India. But one can imagine the figure is very high.

This peculiar bone disease affecting the bony capsule surrounding the inner ear where the bone is the hardest in the human body, invades this area by a sort of softer bone which grows steadily and profusely. The peculiarity is that the choice of site for this growth is the area surrounding the Oval window as a result of which the chief pathology is fixation of the foot-plate of the Stapes to the Oval Window. This, of course, prevents the fine free movement of the Stapes and so conduction of sound waves gets less and less. From 1876 onwards to overcome this common form of deafness, eminent otologists like Kessel - Passow - Barany - Jenkink and others had devised many surgical procedures but all proved unsatisfactory. In 1938 Lempert devised his technique of Fenestration. This gave a great hope but this too had and has its own shortcomings. An advance on this has been worked out recently by Prof. Rosen of Mount Sinai Hospital, New York as a result of which a great hope has dawned in the dark horizon of the deaf. This simple, sincere soldier of peace has travelled over 56 countries at his own expense imparting his technique of Mobilisation of the Stapes to those who were keen to learn. It seems to be a great advance over Lemperts' Fenestration in many ways. I had the unique fortune to be with the master for the full period of his sojourn recently at Madras and Vellore where he operated on a number of cases.

This highly delicate, extremely fine operation requires skilled fingers which a surgeon could develop only after a good number of cadavar work. From my experience of a number of cases Rosen operated here at Madras and from the results of near 100 of my own cases recently operated by me, I feel here is a hope for at least a good percentage of the hard of hearing who had to live a life of allofness, dejection and despondence.

I feel that the advice given by the Master that none should attempt this operation without doing at least 50 operations on the cadavar is a good one because of the delicate area where the manipulation of the Stapes is done and the extremely fine structures involved. It is sad to learn that a few have begun to dabble with this operation without any experience on the cadavar. After doing 50 operations on the cadavar and about 100 operations on the living, I know and feel that none should do this extremely delicate operation without a strong foundation and sufficient experience.

Cases with progressive (acquired) deafness with intact drum membrane and with no history of pus are the proper cases. In many cases there is a family history of deafness. By tuning fork test the air conduction may be markedly diminished but they should have good bone conduction. Proper fine special instruments, a magnifying properly fitting binocular loupe and proper illumination are of great importance for this operation. Most of the instruments should be oxidised dark to overcome reflection which when present is a great handicap for good surgery.

All over the world this recent Rosen operation is being done more and more and it is the duty of every E. N. T. specialist to master the technique of this highly specialised operation so that he can be of greater use to society. The physical disability experienced by the hard of hearing, the psychologic depression reflected in the face of the person, the social aloofness he slowly moves into, the economic repurcussions he might experience due to his inability to hold posts of responsibility on account of this disability go deep into the heart and mind so that quite often these cases come to you with the words, " Sir, there is no use of my living in this world if something cannot be done to help me." Fortunately, it looks that in this "Rosen" operation, lies a hope for quite a large percentage of this unfortunate group of the " hard of hearing."

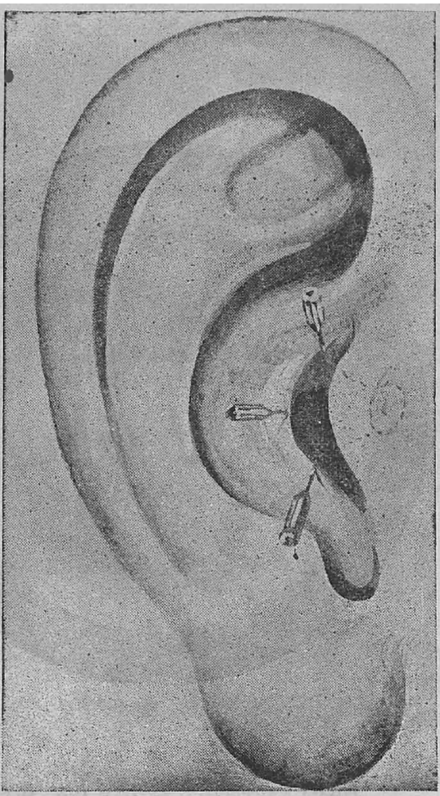

FIG. 1

Injection of ½ c.c., 1% Novacain at four quadrants at the junction of the cartilage and the bone of the external meatus.

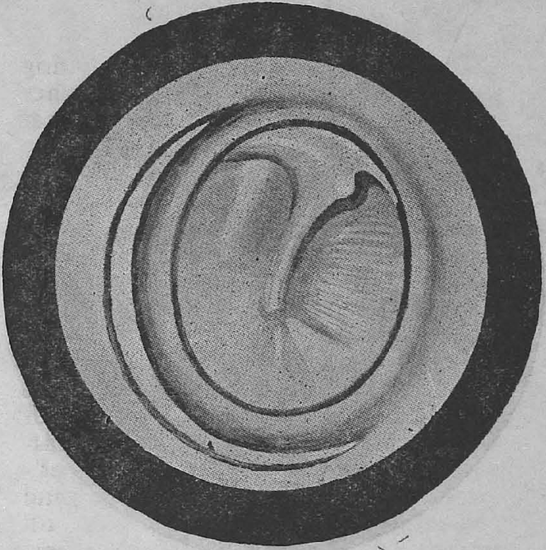

FIG. 2

Incision 6 to 7 mms. from the attachment of drum from 11 'o' clock to 4 'o' clock.

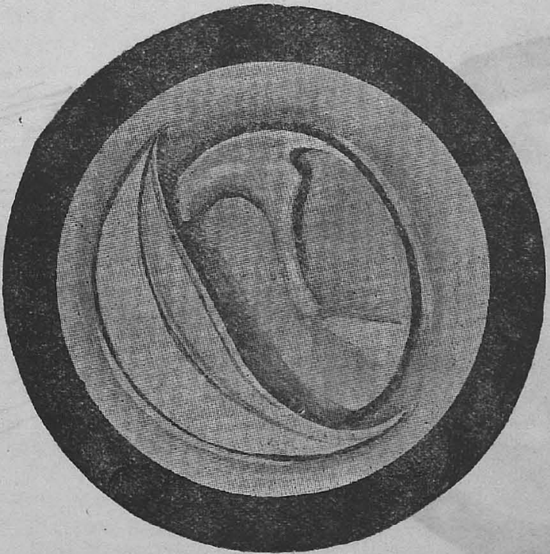

FIG. 3

Seperation of the soft tissues upto the drum membrane.

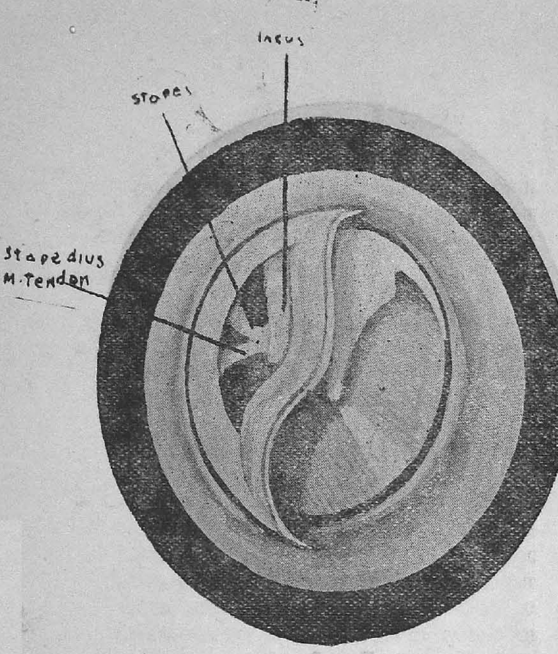

FIG. 4

The drum membrane with the soft tissue is gently lifted forward like shirt-sleeve, exposing Incus, head and neck of Stapes, Stapedius tendon, Round window and Promontary.

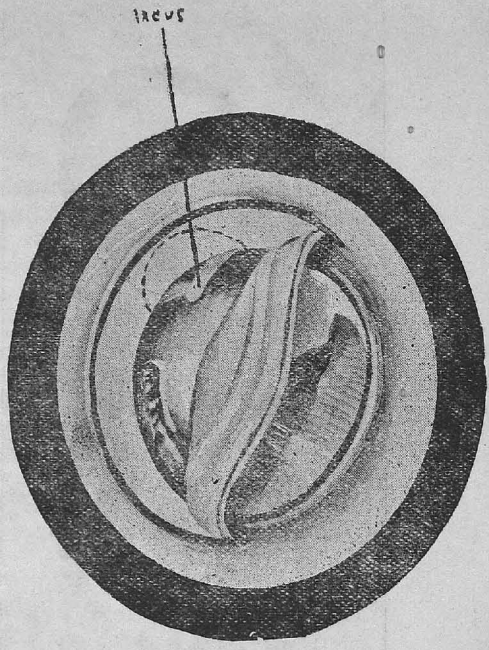

FIG. 5

Incus exposed. Dotted line area to be sliced off.

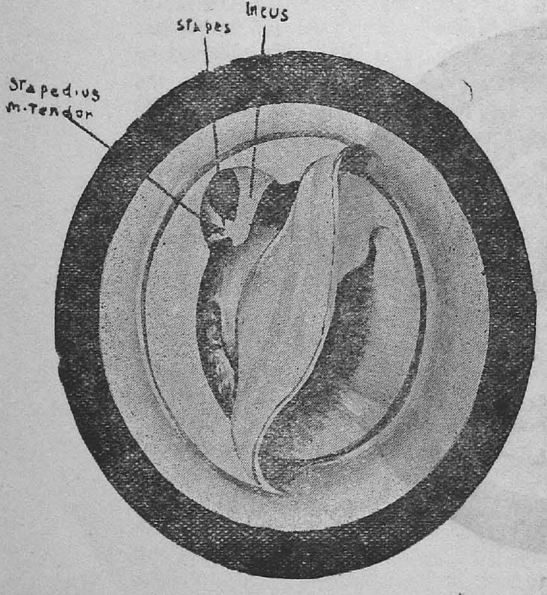

FIG. 6

After slicing off, the bone, more of Incus, Stapes, Stapedius tendon etc. visible.

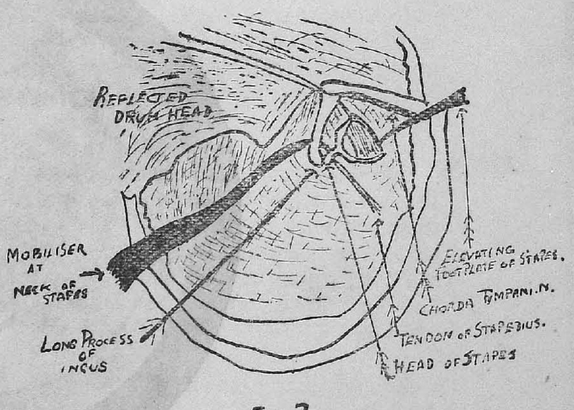

FIG. 7.

In quite a few cases that I have done and the 20 and odd cases of Prof: Rosen I have seen, the expression of happiness and profound gratefulness the patient shows is unequalled to any that a medical man ever will experience.

To have the opportunity to move close to the " hard of hearing " and to feel that you can do something for them to enable them to hear again is indeed one of the most happy feelings, man can ever have.

OPERATION NOTES

The operation is done under local anesthesia. I use 2% novacain to 5 c. c. of which ½ c. c. liquor adrenalin 1 in 1000 is added. Using fine needles ½ c. c. of this solution is injected at each of the four directions medial-lateral-superior and inferior wall of the external auditory canal at the junction of the cartillageous with the bony meatus. This gives perfect anesthesia of the most sensitive drum membrane-the external canal and the middle ear.

1. An incision is put with a special instrument from 11 to 4 'o' clock on the right side and from 1 to 7 'o' clock on the left side, on the bony canal 6 to 7 mms. from the attachment of the drum membrane.

2. With special instruments the soft tissue is carefully dissected down to the drum membrane which with the soft tissue thus dissected is detached very carefully from the Sulcus Tympanicus and lifted out folding on itself forward and upward.

3. By doing this in most cases the Round window and the promontary are visible and often varying lengths of the long process of the Incus and the incudo-stapedial joint. If it is not of sufficient size to manipulate, one has to expose some more of the incus and the incudo-stapedial joint by slicing off the bony meatal margin.

When sufficiently exposed, stapedius tendon - the incudo - stapedial articulation, the head and neck and perhaps the crura of the stapes are also visible. In order to manipulate on the neck of the stapes one uses special instrument anterior to the long process of the incus and the mobilisation is done in the direction of the Stapedial tendon. For this it is necessary to have the incus and the stapes exposed sufficiently by slicing off the bony posterior margin of the external meatus. The mobilisation has to be done extremely carefully - properly and under good lighting arrangements with powerful loupes for proper visualisation.

I am of the opinion that this operation should have the 1st place in rehabilitation of the " hard of hearing ". This has definite advantages over the Fenestration operation in many points. In case the deafness returns after the intial improvement at the 1st operation, as it does in one out of every 5 cases - the operation can be repeated with ease and authorites are agreed on the point that surprisingly enough, for reasons not known, the deafness does not go back to the old level again after such revision. Considering the enormous incidence of Otosclerotic deafness, I feel that it is the duty of every E. N. T. surgeon to master this technique to help the unfortunate-helpless-dejected-depressed group of the " hard of hearing."

In properly selected and successfully operated cases, the return to normal hearing is a wonderful experience, the doctor and the patient have. Let me once again repeat that experience with at least 40 operations on fresh cadavar is of paramount importance before attempting the operation on the living. If this is not done the results will be discouraging and the good name of the operation will be spoiled.

In quite a number of cases, mobilisation of the foot-plate might be impossible on account of strong adhesions and if one uses too strong pressure, the Crura might snap. This is not a happy thing. In such cases what is called direct method has to be adopted.

For this with the special fine dissector one tries to lift up the foot-plate by using the Oval window margin as fulcrum and this has to be done extremely carefully and without hurting the membranous perilymphatic region. Recently I have adopted routine dissection of the foot-plate in almost every case that shows slightest difficulty to mobilise and by constant practice on the cadavar I have been able in many cases to drill a hole in the foot-plate also. The advice of the father of this operation is unfortunately not followed by most here in India, quite a few of whom began to dabble with this finest surgical procedure after seeing Prof: Rosen's few operations without doing any cadavar surgery. Their result was failure. This is not fair for the good name of this surgery or for the author of this operation. After having done over 50 operations on the cadavar and near about 100 operations on the living I know the value of the advice of the author of this operation, who repeatedly said, "do not touch a living case till you finish at least 50 *fresh* cadavar work however great an expert in surgery you may be".

This operation is more difficult and more delicate than Fenestration though the time taken is only 1/6th that of Fenestration. In properly selected cases and properly operated ones the results are extremely gratifying. Proper instruments of proper metallic temper and colour and size are of very great importance towards this end.

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* RHEUMATIC DISEASES IN CHILDREN

WITH SPECIAL REFERENCE TO ITS EFFECT ON THE HEART

by

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RHEUMATIC fever is prevalent all over the world with a natural high incidence in the cold and damper climates. It is more prevalent during the cooler seasons with a preponderance in females, and maximum attacks in children between the ages of 5 and 10. The peak age of onset of infection is 7 years and of rheumatic activity between 11 to 13 years. Less than 1 per cent die during the initial attack and another 25 to 40 per cent die within a course of 20 years. First attack after the age of 20 is rare, but reactivation of previous diathesis and rheumatic carditis can occur at any age.

The cause of this condition is still subjudice. Schlesinger's virus theory has lost its exponents and it is now thought that the primary activation is due to a beta haemolytic streptococcus of group A, but this organism is rarely demonstrated in any lesion even though A S O antibodies are easily demonstrable in the serum of victims. It is also found that antibodies like streptokinase (fibrinolysin) hyalorunidase and desoxyribonuclease have been found. These could easily explain the many manifestations in the pathology of rheumatism. The effusions into the joints, the rheumatic nodules and the fibrous replacement thereafter and the adherent scars produced in the internal organs are mainly due to these various antibodies. A rising titre of these antibodies shows a recent infection. Anti M precipitins are also abundant with an excessive gamma globulin. Thus we think this manifestation is due to an allergic response, post-streptococcal. The hereditary theory of autosomal recessive gene has not found many followers. But in actual practice there is some hereditary diathesis for rheumatism occuring in the siblings. Finally, it is now assumed that repeated attacks of haemolytic streptococcal infection of the tonsils, suppurative complications, lymphadenitis etc. is followed by a pyrexial reaction with its other manifestations and later by the action of the allergants and the toxins synergistically on the mesenchymal tissues to produce the rheumatic syndrome. It is an abnormal response to infection and this may also be added under the diseases of mal-adaptation.

The more modern view is that the rheumatic syndrome is one of collagen diseases since the collagen matrix bears the brunt of this attack. Practically we find the rheumatic manifestation in the various organs that contain mesenchymal tissues and not so readily in others which are sparse in this type of tissues, for example, in the brain, the mesenchymal tissue is found only in the vascular bed and the only cerebral complication that we meet with is an encephalitis with a special preference for the highly vascular basal ganglion regions like the globus pallida resulting in the well-known chorea. Thus the above infection, its predilection for mesenchymal tissue, its production of antibodies mentioned, easily explain the pathology we meet with and the symptomatology that derives therefrom.

The clinical features of rheumatic fever are well known and one should just

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touch upon that only, but before doing so I wish to stress the importance of recognising earlier stages than the actual fever. One should be able to make out the rheumatic state and take measures to combat it and to stop the march of events. This state consists of five phases. Phase 1: repeated upper respiratory tract infections due to haemolytis streptococcus like pharyngitis, tonsillitis, otitis and scarlet fever. This is a stage of focal infections. Phase 2: a period of latency 4 to 6 weeks or more after phase 1 when the child develops hypersensitivity —the child tires easily, has vague limb pains, may manifest erythema nodosum with a raised E. S. R. etc. Phase 3: this is a period of clinical rheumatic fever with migrating polyarthritis, monocyclic, polycyclic or continuous, fever, tachycardia, the presence of rheumatic nodules, erythema modosum, erythema marginatum and finally carditis. This is accompanied by leucocytosis, anaemia and raised E. S. R. This phase may be acute, subacute or insidious. Sometimes it may not be pyrexial, but simple choreal manifestations or severe growing pains. (Here I may be permitted to say that the vague limb pains in an asthenic child which one may meet with at the end of the day but which disappears with good sleep by early morning is not pathological, but if the child wakes up after a good night's rest with joint or muscle pains one could be reasonally sure that it is a rheumatic state.) Phase 4: period of rheumatic inactivity or quiescence. This period is likely to be reactivated by trigger mechanisms like upper respiratory tract infections. Phase 5: is a period of consequences or complications. As outlined before, the complications arise in about 50 to 60 per cent of the rheumatic patients. It may be mild, moderate or severe. This depends upon the severity of the original disease, the number of recurrences that have occured and to what extent treatment by way of rest has been given. The main brunt falls upon the heart with minor complications elsewhere, like chorea, refractory anaemia, pneumonitis, cerebral arteritis and mental disturbances. Further, superinfection in rheumatic heart by streptococcus viridans is common, resulting in subacute bacterial endocarditis.

Among the cardinal signs in the first three stages and specially the third are. (1) a tachycardia uninfluenced by rest, (2) rarely bradycardia and extrasystoles, (3) a raised E. S. R. with a hyperglobulinaemia and positive precipitin test mentioned before and (4) auscultatory changes in the heart sounds with varying murmurs (5) changes in the E. C. G. with a prolonged P. R. interval and rarely other types of heart blocks. These are in addition to the commonly noted symptoms like excessive sweating with a peculiar musty odour and the joint and skin changes already referred to. The presence of rheumatic subcutaneous nodules means a very severe infection and the cardiac damage will be gross. Thus there is a steady onward march in the cardiac complications ending in a cardiac cripple and I shall discuss this next and take up the efficacy of treatment or otherwise and preventing them last.

Carditis or rheumatic heart disease is a natural sequel. The heart is invariably affected in some way or other, mild, moderate or severe. It may be a simple tachycardia or bradycardia, failure of the heart, pericarditis with or without effusion, endocarditis resulting in valvular changes, severe myocarditis or a combination of all. The seriousness of this stage can be visualised by statistics. In the United States, 60% of all the heart diseases are due to rheumatism. Duchett and Jones studied 1000 rheumatic heart cases for 10 years and found more than 25% mortality in them in that area, and in the remainder 50% were left with cardiac damage. Cohn and Levine studied 3000 cases for 10 years and said that half died in nine years' time and majority of the remainder were lost within 13 years. This shows us the tragic end of rheumatic patients.

Now rheumatic endocarditis can be suspected early in the patient by the prolongation of the first sound and the diminution in its intensity. A very early sign may be a soft blowing systolic murmur. A transient mid-diastolic murmur (Carey Coombs) may indicate no permanent damage, but should be always regarded seriously. As the rheumatic pathology advances, adhesions in the cusps of the various valves take place. The commonest affected is the mitral resulting early in incompetence and later stenosis becomes the overwhelming factor. Aortic stenosis is rarer and affection of the pulmonary valve uncommon. But one often meets with a polyvalvular syndrome of the mitral and aortic valves, Mitral stenosis may be moderate or severe, the valve may be pliant or calcareous. It may be accompanied by mitral regurgitation which generally leads to giant left atrium. The sequenee of events could be easily arranged as (a) Mid-diastolic murmur with or without a thrill and a pliant valve. An opening snap can be often heard at this stage. (b) Advancing sclerosis of the valve with an added presystolic element, crescendo in nature. There is a definite apical diastolic and presystolic thrill. (c) Enlargement of the left atrium and pulmonary conus, pulmonary hypertension with pulsating vessels in the pulmonary window when screened. (d) If accompanied by regurgitation, the resultant giant atrium is seen as a shadow through the cardiac shadow in the skiagram. (e) Pulmonary oedema and pulmonary hypertension slowly leading to rightsided failure. There can be a pure left auricular failure in this condition. Auricular fibrillation is very common, and once this sets in the presystolic murmur if present naturally disappears. Embolic episodes can occur in the various organs inclusive of the central nervous system and leads to further deterioration. Electrocardiographically, in advanced mitral stenosis of the pure type there is left auricular hypertrophy as shown by the notched P wave known as the F. mitrale of Paul Wood. Later on when the pulmonary hypertension increases and the right ventricle and auricle become dilated the notching in the P wave disappears. We all know that a right auricular hypertrophy is shown by a tall P wave in the E C G.

Thus in the march of symptomatic events, a gradual loss of cardiac reserve a lessening of the exercise tolerance leads on to the inevitable unless the case is favourable for surgical intervention when palliative surgery could be done. Mitral stenosis of moderate degree is compatible with life depending on its type upto thirty years or more, but has a rapid deterioration in women during the child-bearing age and it is indeed a landslide then.

Aortic stenosis:

Aortic stenosis on the other hand is rarer but has far-reaching effects. One could easily imagine an enlargement of the left ventricle, a generalised systemic ischaemia and sudden loss of consciousness, anginal syndromes due to defective filling in the coronary vessels and resultant cardiac ischaemia. Sudden death or left ventricular failure with pulmonary oedema is the outcome. Surgery is indicated here when life becomes intolerable if there is no accompanying regurgitation of the valve.

Mitral incompetence on the other hand is common but of less import unless it is gross. Systolic murmurs of the mitral area are quite common in rheumatic patients early in its onset due to dilatation of the valvular ring as a part of generalised myocarditis. This systolic murmur generally loses its intensity as the patient recovers but may continue as a soft blowing grade I or II type of murmur throughout life. It may be musical and does not shorten the life or the activity of the individual, This type of systolic murmur becomes a problem to the practitioner and more so for life

insurance. To restrict the activity of the child on the basis of this murmur or not is a hard one to decide. But one should be guided by the other factors involved, namely X-Ray changes, ECG, ESR, pulse rate and most important the cardiac reserve. If they are normal as in many cases they are, this grade I or systolic murmur can be ignored. Here a word of caution is necessary since it has been found in autopsy that the fibrosed valve still shows rheumatic activity in its core, many year after the mitral infection.

Pericarditis :

It is not uncommon during the febrile periods, but when the temperature has touched normal and repeated recrudescences of pyrexia occur accompanied by restlessness. pallor, and precordial or substernal pain it should make one suspect this condition. An effusion develops often suddenly, the pericardial rub easily heard earlier disappears with muffling of heart sounds. If cardiac tamponade is present, pulsus paradoxus is seen. But pulsus paradoxus is not so common or so well felt as in constrictive pericarditis. Skiagram is very helpful to diagnose this condition by the globular nature of the cardiac shadow with a widening in its base and change of shape with change of position can also be demonstrated. E C G shows the typical pericarditis changes with a low voltage, raised R S T segment and inverted T. This E C G has no Q wave which differentiates it from what of myocardial damage.

Myocarditis :

This is an everpresent feature in rheumatic fever in some degree or other. It is always present in all the cardiac fever complications described and more so in pericarditis. Myocarditis is a very dangerous condition, and is the cause of death in acute rheumatic fever when it does occur. Undue tachycardia, soft first sound, increasing enlargement of the heart with soft systolic murmur, abnormalities in the E. C. G. like prolonged P. R. interval and Q. T. interval, various ranges of heart block may also be evident. Rheumatic myocarditis is often killing and more rapidly than other conditions.

It is important to know the active and inactive or burnt out types of rheumatic diseases of the heart. Fever, tachycardia, anaemia, changing cardiac signs, failure to gain weight, raised E. S. R., abnormalities in the E. C. G. all show active or smouldering condition. Activity implies fresh and increasing damage to the heart, fresh laying of fibrous tissue, fresh or more contraction of the valves, stenosing them more thereby. A modern indicator of help regarding rheumatic activities is the estimation of C. reactive protein precipitins and is considered to be a delicate one.

The other complications like chorea, kidney damage, persistant loss of weight are not alarming, but the anaemia that follows rheumatic fever is often resistent to treatment by the ordinary methods. Exhibition of iron by the intravenous or intramuscular route is often indicated.

Treatment :

Prophylaxis or prevention of rheumatic fever is the ideal one. In all susceptible children of the age group between 5 and 8 who are necessarily school-going in character and who have shown a tendency to streptococcal infection like tonsillities, lymphadenitis, sore-throat chromic rhinitis and otitis media, early institution of prophylaxis could prevent the sensitisation of the child to the various toxins of this organism, thus preventing the onward march into rheumatic stage. Such infections should be adequately treated and these children should be guarded against chills. During the cooler months of the year prophylaxis can be given with either sulpha drugs or oral penicillin, the latter being a better selection. A recovered rheumatic child should avoid contact

with further sources of streptococcal infection, and in kindergarden schools use of common dishes, towels etc. should be avoided. One of the potent sources of lurking infection is the tonsil. Tonsils if badly affected with pockets of pus should be removed. But milder grades can be easily overcome by the modern antibiotics.

Treatment the acute phase :

There is nothing much that I can add to what is being practised everywhere. The primary thing is bed rest and it should be absolute. Knowing well the pathology of this disease in producing gross myocarditis, one should emphasize on rest again and again. Rest, apart from the heart, is alleviating to the painful joint condition too. Warmth to the joints by any means at our disposal, giving of large amount of fluids to replace the loss of water sustained by sweating, by looking occasionally at the electrolyte balance in the individual and replacement if necessary, and giving of good nourishing food is more than half that is to be done. The drugs used are some specific and others non-specific, the term specific being only of relative character. Vitamin C in very high doses is of great help during the exudative phase of this disease. Sodium Salycilas and Aspirin are still the sheet anchor of treatment with drugs. Sodium salicylas or aspirin must be given at very high doses, the blood salicylate level must be kept near 30%, a workable formula being giving of Sodium salycilas 1 gram for 7 kilogrammes of body weight till signs of early toxicity like ringing in the ears, nausea and vomiting, hurried breathing comes on and reducing the dosage thereon a little. Salycilates produce a reduction in the water metabolism, stimulate protein katabolism and aggravate respiratory alkalosis by hyper ventilation. All this has dehydrating action on the joints with relief of pain and a fall in the E. S. R. If there is persistent salicylism, bicarbonate may be given in small doses.

I prefer to use in cases of rheumatic fever high doses of Aspirin, plain or as calsaspirin or dispirin. For an average Indian child of 50 to 60 pounds aspirin can be safely given in 10 grain doses three times a day. It is supportive to the heart, the only drawback being aspirin must be given in a full stomach as it is likely to produce gastric irritation. We have treated in our hospital many cases with a far higher dosage of Aspirin. While under this therapy as with salicylas, one should watch out for thrombocytopenia with the resultant haemorrhages, petechial and elsewhere. A daily examination of the urine for RBCs is of value here.

Recently it has been said that salicylic molecule acts like cortisone since it has got a chelating ring incorporated in it.

Cortisone as all of us know has been extensively used in acute rheumatic fever. It acts in two parts on the exudative and proliferative reactions of the disease by suppressing them. ACTH acts on the exudative phase more than on the proliferative phase. These hormones do shorten the acute phase, relieve the signs and symptoms, regress the ESR and sometimes even the early murmurs of a rheumatic disease (which I have already pointed out may do so of their own accord.) These hormones should be exhibited with caution as any lurking infection can flare out and an antibiotic cover like penicillin is indicated. There should be only a tapered withdrawal of these drugs.

Cardiac failure occuring in the acute phase is not so responsive to digitalis being a toxic myocarditis. Convalescence of the acute cases should be prolonged and supervised and bed rest is necessary till the heart rate is slow during sleep, ESR is normal and there is no leucocytosis. Gradual getting up should be encouraged, graduated exercises without undue fatigue and this can be regulated

by the pulse. In an ordinary case of pyrexia of rheumatic origin becoming normal in about a fortnight the rest should be three times that. This test is probably the one that prevents further cardiac damage. None of the drugs given above prevents the cardiac damage but only alleviates the patient and rest alone minimises the resultant cardiac deficiency.

Post-rheumatic Cardiac Patients :

Once the rheumatic state is arrested cardiac damage is not progressive provided there is no recurrence or recrudescence of the complaint. The presence or absence of continuing rheumatic activity should be estimated as previously mentioned. Once it has reached the burnt out stage and the final assessment of the damage is made proper advise should be given in the following lines: (*a*) Prophylaxis which is already explained. (*b*) The necessity to live within the limited cardiac reserve and restricting the child's movements should be considered. An odd systolic murmur with no other changes may be ignored with some impunity, but a mitral stenotic murmur should be given the respect it deserves. Not going into the treatment of cardic failure occuring hereafter or of auricular fibrillation or of embolic episodes, I will briefly touch on the prognosis of mitral stenosis from childhood on to adult life. It is an accepted fact that mitral patients have a restricted life span, but it is highly narrow in the case of women. All the milestones of life puberty, adolescence, youth or womanhood weigh heavily upon these hearts. Each landmark reduces the life span by a few years. Thus naturally man is at an advantage since the poor woman marries and has to undergo child-birth. The drain upon the reserve of the heart during pregnancy and child-birth is immense and by about the third pregnancy most if not all of the mitral cases reach the end. Thus heart disease in pregnancy is a problem and it should be explained to them that they may pass through pregnancy safely, with a caesarian section, but thay have a greater strain in looking after the newborn child. The period of greatest danger are the 7th and 8th months of pregnancy and the first two days after delivery and more so if there is no help at home to look after the new born child. Each prgenancy increases the cardiac deficiency permanently. Thus they should be advised to have two children since one is unsatisfactory, shepherded through pregnancy well, given domestic aid and sterilised promptly after the second child. This also depends upon the original state of the heart before embarking on pregnancy.

The place of surgery in mitral stenosis is limited. There are many contraindications like (1) gross incompetence of the mitral or the aortic valve (2) polyvalvular lesions (3) giant left atrium (4) repeated attacks of congestive failure (5) old age. Having got over these stumbles which very few can, surgery can be thought of. Surgery is simple and in expert hands takes about half an hour to fourty-five minutes for the completion of the operation and the hazards are fewer than abdominal surgery. As such, selected cases should be sent up for surgery now that we have many centres in our country doing this type of surgery since it converts a destined cripple to normalcy at least for some years. Surgery is indicated and is a crying need in (1) recurring attacks of pulmonary oedema (2) sudden deterioration in exercise tolerance (3) incipient failure of the heart and (4) in women during pregnancy or even after delivery. Assessment of cases for surgery is made by clinical findings, radiological evidence, E. C. G. changes and if necessary cardiac catheterisation.

Thus I have come to the close of this chapter of rheumatic diseases of the heart and I may be permitted to say that prevention that is prophylaxis is better than cure of this disease.

CASE NOTES

Myeloid Leukaemia

Mrs. A., female aged 30 years was admitted with the complaint of a swelling over the left side of the abdomen, occasional fever and marked weakness — all of six months duration.

On examination the patient was found to be an emaciated, anaemic individual with an enlarged spleen extending down into the pelvis and with an enlarged liver extending four inches below the costal margin. There was no localised or generalised adenopathy.

Laboratory findings:

R. B. C. count: 1·5 mill/c.mm.

Haemoglobin: 45 per cent.

Total W.B.C. count: 2,60,200/c.m.m.

Differential count: Polymorphs 48%, Lymphocytes 28%, Eosinophiles 2% and Monocytes 26%.

Blood picture: Increased number of neutrophiles and myelocytes with occasional myeloblasts.

Sternal puncture: Typical picture of Myeloid leukaemia.

The above case of Chronic Myeloid Leukaemia was treated with Myleran with considerable relief.

Discussion:

It is not unusual to see cases of Myeloid leukaemia. Patients come rather at advanced stages when the large spleen becomes a discomfort. It is also known that splenic enlargement attains its maximum in this condition. Chronic myeloid leukaemia is not necessarily fatal immediately, but has a prolonged course. Modern treatment has been successful in prolonging life even upto twelve years after diagnosis. The main points in treatment are

(*a*) Reduction in the size of the spleen to tolerable proportions.

(*b*) Reduction in the number of abnormal cells in the peripheral circulation.

(*c*) Reduction in the various bleeding episodes that can occur due to abnormal drop in the thrombocytes.

(*d*) Overcoming of infection.

Various measures such as Deep X-Ray therapy, urethane, nitrogen mustard, Radio-phosphorus, splenectomy and Myleran have been tried. But the drug of choice is Myleran which is controlled by periodical day to day or weekly leucocyte counts. Optimum dosage appears to average 0·06 mgm per kilogram body weight per day by mouth. With larger doses thrombocytopenia may occur. Myleran treatments does produce reduction in the number of abnormal leucocytes as well as in the size of the spleen. Blood transfusion and other haematinic factors to overcome the concomitant anaemia are additional measures. Antibiotic cover is essential to combat even minor infections and Penicillin is the drug of choice as it has no known depressive effect on the bone marrow. In the case under discussion, Myleran afforded cosiderable relief to the patient.

Intervertebral Disc Prolapse

Mr. S., aged 37 years, a subject of low backache for ten months was admitted with the complaint of inability to walk properly and excruciating pain over the back shooting down the right thigh to the back of the right ankle. The pain used to become intense during coughing and sneezing. Severe pain in the right gluteal region made it very difficult for

With kind permission from the North Arcot District Clinical Society.

him to sit on any hard stool or chair. During the past three months, the pain had become intolerable not allowing him to sleep and he had to take daily one or two injections of Novalgin for the relief of the pain. There was no history of any accident or sudden strain to his back.

On examination he was found to be a well-nourished individual walking with a limp on the left side dragging the right leg painfully and afraid to sit down. There was no tenderness over the spine, but a tender spot was noticed in the right gluteal region over sciatic nerve. There was also certain amount of wasting of the right glutei and the right quadriceps. The right ankle jerk was absent but other reflexes were normal. There was complete loss of sensation over the distribution of the first sacral nerve. The straight leg raising test was positive. i. e. after flexion of the hip, the right leg could not be straightened out beyond the right angle.

A diagnosis of prolapse of the Intervertebral disc was made and he was treated conservatively with extension. The spine was kept in extension by raising the foot of the bed by nine inches and by traction from the iliac crest with a weight of ten pounds. The patient showed remarkable improvement by the second week of traction. By the end of the third week, he had no pain at all and the straight leg raising test was normal.

Discussion :

The Intervertebral disc consists of three different components ;

(*a*) The hyaline cartilaginous plates above and below.

(*b*) The nucleus pulposus which is a semigelatinous mucoid material enclosed in a cartilaginous capsule.

(*c*) Annulus fibrosus consisting of a series of concentric lamellae of fibrous tissue surrounding the nucleus.

The nucleus pulposus is normally under elastic tension and acts as a shock absorber. Alteration of the water content of the nucleus pulposus or wear and tear changes in the annulus fibrosus results in protrusion or prolapse of the nucleus pulposus The prolapsed material is usually covered by a thin layer of annulus fibrosus and is commonly a little to the side of the midline, lateral to the posterior longitudinal ligament, interfering with the neighbouring nerve roots due to compression, stretching, friction or degeneration or a combination of any of these factors. Secondary changes in the adjacent bony components also may play a part. The commonest site at which the prolapse occurs is between the fifth lumbar and the first sacral vertebrae. Minor repeated trauma or a sudden injury may predispose to this condition.

The clinical features are characteristic as described in the case under discussion. Additional investigation to confirm the diagnosis are X-Rays, myelography and in some cases examination of the Cerebro spinal fluid.

Treatment: The natural history of a prolapsed disc is a process of healing with fibrosis and shrinking of the prolapsed material. The vast majority of cases recover from the symptoms by conservative treatment.

Conservative treatment :

(1) Absolute rest in bed with skin traction to keep the spine in extension. This regime dramatically improves the condition especially in acute stages and the rest should be continued for two or three weeks. In the case under review this was the method of treatment adopted with gratiyfying results.

(2) Spinal muscle extension exercises. Patients are warned not to do any flexion movement or lift any weights.

(3) Epidural injection of 100 - 200 c.c. of saline has been advocated, but this is not very successful.

Surgical treatment :

Though 90% of cases recover with conservative measures, surgery is indicated when,

(1) there are frequently recurring acute attacks not responding to adequate conservative treatment.

(2) there is a massive prolapse with cauda equina pressure.

(3) there is severe interference with root function causing weakness of muscles and sensory changes.

The disc may be removed by (a) formal laminectomy and intradural approach or (b) by an interlaminar approach. In some cases spinal fusion also is indicated.

Sub-acute Combined Degeneration

Patient S, male aged 24 years was admitted with the complaint of inability to use his lower extremities freely for the last two years. The previous history of the patient revealed that in 1949 he had an attack of pulmonary tuberculosis and in 1954, an attack or weeping eczema on his legs and feet. For the latter condition he was treated by some native measures including multiple incisions to let out his 'bad blood'. While suffering from eczema the patient noticed certain amount of loss of superficial and deep sensations over his legs. Later his gait became unsteady necessitating the use of a stick. He had also been treated in the Madras General Hospital for this complaint.

On examination, the patient was found to be an anaemic, moderately nourished individual walking with a paraplegic, ataxic gait. His central nervous system and upper extremities were normal. Fundus examination showed certain amount of temporal pallor. The lower extremities were spastic on both sides with some amount of atrophy and impairment of the muscle tone and power. The deep and superficial reflexes on either side were exaggerated and the planter reflexes were extensor on both sides. The visceral reflexes were normal. Romberg's sign was positive. Sensations of light touch and pinprick were impaired on both sides from the L I level. Temperature, vibration and joint senses were also impaired on both sides. There were no trophic changes.

Investigations :

Blood pressure : 100/70 m. m.

Urine : No abnormalities seen.

Blood : R. B. C. count : 37 millions/c m. m.

Colour Index : I

W. B. C. count : 7600/c.m.m.

Differential count : Polymorphs 70, Lymphocytes 18 Eosinophiles 12.

Blood picture : Dimorphic with few macrocytes and no nucleated R. B. Cs.

Sternal puncture : Megaloblastic reaction.

Lumbar puncture : C. S. F. clear under tension. Cells : 4/c. m. m. Quickenstedt's test : No block.

Fractional Test Meal : No free Hydrochloric acid.

After Histamine : No free Hydrochloric acid.

Discussion :

Subacute combined degeneration is a progressive disease usually associated

with pernicious anaemia in which the white matter of the spinal cord degenerates, the incidence of the lesions being particularly on the posterior and lateral columns. We now know that the postero lateral sclerosis can occur not only in pernicious anaemia but also in other deficiency syndromes. Pernicious Anaemia with Subacute combined degeneration is supposed to be a rare condition in Indian climates. The above case illustrates by all its findings that the posterolateral sclerosis has taken place subsequent to a true pernicious anaemia. This case was treated with Vitamin B 12 etc. and a follow up for six months showed remarkable recovery in the patient's ambulation.

Cortisone in Typhoid.

The case under discussion was one of typhoid fever of an extremely toxic type. The patient had been having continuous fever for 12 days and he had become very toxic and restless. In spite of intramuscular Chloromcetin therapy for 4 days, the toxicity had not abated patient had loose motions and the general condition became very low. On the 15th day, in addition to chlormycetin, Cortisone also was given, 25 mgms every 6 hours. In two days the temperature became normal the general condition improved and the patient lost his toxic facies. The Cortisone was stopped, but chloromycetin was continued. On the next day the temperature again mounted up and signs of toxicity reappeared. So cortisone was given for one more day and very soon the patient became normal.

In this particular case, Cortisone had a dramatic action and saved the toxic patient from a fatal termination. The use of cortisone in such a toxic condition must be very cautious and the drug must be exhibited only for a brief period, remembering to continue the specific antibiotic therapy as usual in spite of the addition of Cortisone.

Mal-absorption Syndrome.

Mrs. N., aged 36 years was admitted on 28-4-56 with the following complaints of one year's duration :— Ulceration of mouth, loss of weight, loss of appetite, loose motions and weakness.

One examination she was found to be a moderately nourished, anaemic female with a raw tongue and a soft flabby abdomen. Liver and spleen were not enlarged and other systems were normal.

Investigations :

Urine : Normal.

Stools : Pale, bulky and semisolid with plenty of strach granules. fatty globules, R. B. C.s and Ascaris ova.

Blood :
- Blood Pressure : 20/80 m. m.
- Haemoglobin : 50 per cent.
- Differential Count : Polymorphs 61, Lymphocytes 37 Eosinophiles 2.
- E. S. R. 1st hour : 21 m. m.
- 2nd hour : 53 m. m.
- Blood picture : Presence of macrocytes, poikilocytes and leukopaenia.
- Sternal puncture : Megaloblastic.
- Fractional Test Meal and Sputum : Normal
- Dried Faecrs : 35% fat.

Discussion :

Mal-absorption eyndrome is a defective absorption of one or more of the necessary foodstuffs, mineral salts or vitamins. These get associated with abdominal distension, loss of weight, diarrhoea, anaemia, tetany etc. These were being passed in the past as steatorrhoea since fat metabolism has been noticed to be upset, Mal-absorption can be caused by lack of absorbing surface of the bowel as a result of short-circuiting operations, extensive resection of the gut or its destruction by disease. Even intestinal hurry and post-gastrectomy symptoms would result in this. Rarely changes are due to bacterial flora symbiotic in character which may vitiate the absorbing surface. The absorbing surface can also be affected by diseases of the wall of the intestine wherein lacteals and intestinal villi are destroyed. Contributory foctors may be lack of bile which is necessary for the emulsification of fat and its adsorption or lack of pancreatic digestive emzymes which will produce unsaturated acids which act as irritants producing intestial hurry. Intestinal allergy due to gluten in wheat may produce coeliac disease. Thus the conditions are tropical sprue, idiopathic steattorrhoca and coeliac disease. These diseases are characterised by pale, bulky greasy stools with normally split fat thought it may be in excess. Total fat content of the faeces may be more than 25% of the dried specimen which by itself is diagnostic. A Glucose tolerance test is flat and aminoacid absorption if affected may produce wasting of muscles. Calcium absorption is deficient due to changes in the PH of the intestine resulting in rickets, dwarfism, osteomalacia, pathological ftactures etc. Hypocalcaemia leads to tetany and vitamin K lack produces hypoprothrombinaemia or haemorrhagic states. Deficiency of the B complex group is shown by a red angry tongue, angular stomatitls and peripheral neuritis. B complex deficiency occurs due to lack of absorption or lack of intake or due to lack of endogenous production by the intestinal flora.

Anaemia may be dimorphic in character. X-Ray of the bowel after Barium gives a deficiency pattern due to flocculation of the Barium due to excess of the mucus. In diagnosis, this condition has to be differentiated from ulcerative colitis. Crohn's disease and Tubercular enterocolitis. The treatment devolves on diet wherein carbohdrate is taken in reduced quantity with folic acid and calcium thrown in plenty. Supplementation with Vitamin D and Vitamin K and B complex group of drugs and parenteral Iron therapy are also indicated. Cortisone has very little place in this condition in contradistinctiou to its great role in ulcerative colitis. Diet should be emphasised again to be important as high protein, low carbohydrate, and low fat ln character.

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Homatropine Methyl Bro.		0·75 mg.

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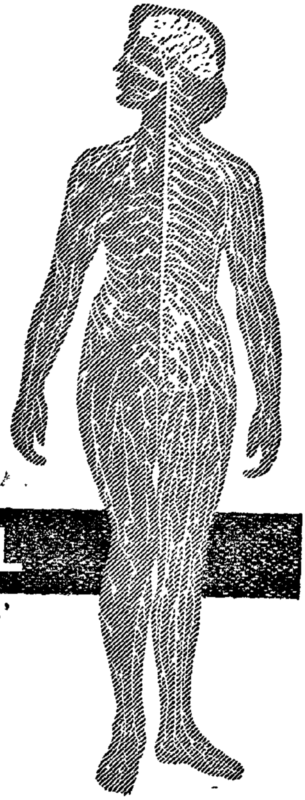

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INDIAN MEDICAL ASSOCIATION
MADRAS STATE BRANCH

Extracts from the minutes of the meeting of the Council of the Madras State Branch of the Indian Medical Association held at the "Association Hall". Government Head-quarters Hospital, Kozhikkode, on Saturday the 22nd June 1957 at 3 p.m.

Dr. Y. P. Vasudevan presided.

12 MEMBERS WERE PRESENT:

1. 28 members have sent messages expressing their inability to attend the State Council Meeting:

2. **Condolence Resolution**: Resolution condoling the death of Dr. T. S. Shetty M.D., of Pudukottah and Dr. T. V. Muthukrishna Iyer of Nagapattinam, was moved from the Chair and passed unanimously, all the members standing for a minute.

3. Statement of Accounts for the month of March, April and May 1957 have been approved.

4. South Indian Branch of the Brltish Medical Association—Funds relating to —share due to the Madras State Branch, I. M. A.—Request of the Central Office for donation of the amount to Central Office Building fund—consideration of:

The Council, after a short discussion, decided to donate its share of the funds, whatever it may be, to the Building Fund of the Central Office with the suggestion that the actual amount of the donation may be specified as the contribution of the Madras State Branch, I. M. A. towards the Central Office Building Fund in response to the appeal for funds.

Hospital Stoppage—levy of concession to medical officers in the Municipal Sevice on a pay of Rs. 300 and over—need to amend the rules on the subject—consideration of:

RESOLUTION

"Whereas the rules governing medical attendance and levy of fees in Government Medical Institutions in the State of Madras as revised and brought uptodate and published in G. O. No. 2800, Health, dated 24th September 1956, do not contain any provision for Medical Officers in Municipal Service drawing a salary of Rs. 300/00 and above; Whereas Note (3) to G. O. No. 2053, Health, dated 29—5—1953 mentioned on page 4 of the Rules for Medical Attendance published in G. O. No. 2800, Health, dated 24-9-56 specifically provides that all Municipal Commissioners and District Board servants drawing a salary exceeding Rs. 300 per mensem whether they are State Government servants or not will be treated as though they are Gazetted State Government Servants for purposes of medical concessions at State Government Hospitals inclusive of medical attendance; Whereas Medical Officers in Municipal Service in certain Municipalities in the State who draw a salary of Rs. 300/00 and above are not covered by the provision in Note (3) referred to supra and their position is anamolus so far as the application of Rules for Medical Attendance is concerned in that they are not treated as Gazetted Officers due to the exclusion of the category of officers in the Note (3) and they cannot be treated as non-gazetted servants by reason of the salary limit; and Whereas it is desirable that Medical Officers in Municipal Service who draw a salary of Rs. 300/00 and above should be treated on a par with Municipal Commissioners and District Board servants referred to in Note (3) to G. O. No. 2053, Health, dated 29—5—1953 in the interests of equity and justice; this Council of the Madras State Branch of the Indian Medical Association requests the Government of Madras to kindly take the needed action to amend the G. O. No.

2053, Health, dated 29—5—1953 in a suitable manner so as to bring Medical Officers in Municipal Service drawing a salary of Rs. 300 and above also in the purview of Note (3) to the said G. O."

Dr. M.G. Nair, M.B., B.S., TDD (M)., TDD (W)., of Coimbatore was nominated as a delegate for the 11th General Assembly of the World Medical Association to be held at Istanbul, Turkey, from 29—9—1957 to 5—10–1957.

New Members–supply of journals with back numbers—complaint of Tanjore Branch, I. M. A.,—letter from the Central Office on the subject—consideration of:

The Hony. General Secretary, I. M. A. Central Office decided that back copies of the journal need not be supplied to members enrolled at any time during Association Year, and that therefore it is not obligatory to supply the new members with all the back copies of the journal, but that the Journal Office will always try to supply them if the copies are available.

Members of the medical profession and Rent Control Act—complaint from the Hon. Secretary informed the house that complaints of the matter may, with advantage, be considered at the open session of the ensuring 12th Madras State Medical Conference and suitable decision arrived at.

Medical Examiners in the Life Insurance Corporation—reinstatement of affected members of I. M. A.—representation received from two members—consideration of: The Zonal Manager has called for applications from the two members for considering their appointment as authorised Medical Examiners, and hoped that they would succeed.

Madras Medical Service—Extension of age limit of medical officers or re-employment of medical officers in the teaching and General line—Orders of Government — consideration of; The Hony. Secretary placed before the House of a copy of G. O. Ms. No. 1529, Health, dated 20—5—1957 on the subject and the Council recorded the G. O.

The Twelfth Madras State Medical Conference, 1957—Venue and dates—decision of: decided to accept the invitation of the Coimbatore Branch, I. M. A. and fixed the 23rd and 24th of November 1957 as the dates on which the Conference may be held at Coimbatore.

Employee's State Insurance Corporation—extension of benefits to the families of workers—capitation fees—statement and notes of Dr. Chamanlal M. Mehta, representative of I. M. A.—confidential circular from Central Office:

RESOLUTION

" This Council of the Madras State Branch of the Indian Medical Association expresses its disapproval at the existing separate scale of fees for various Specialists under the E. S. I Scheme and requests the introduction of a uniform rate of remuneration for all the Specialists employed under the E. S. I. Scheme as varying rates of remuneration will tend to create invidious distinction among the various Specialists which is not desirable."

Circulars of the Indian Medical Association, Central Office were read and recorded.

Circulars of the Indian Medical Association, Madras State Branch were read and recorded.

Vote of Thanks:

RESOLUTION

"This Council expresses its sincere thanks to the Malabar Branch of the Indian Medical Association for the excellent arrangements made for the successful conduct of the meeting of the State Council of the Madras State Branch, I. M. A and for the fine arrangements made for the boarding and lodging of the visiting members of the State Council."

The meeting terminated at 5 P. M.

Y. P. VASUDEVAN, *President.* C. ARUMUGAM, *Hony. Secretary.*

ASSOCIATION NEWS

CHINGLEPET BRANCH

The Annual meeting of the Branch was held on Saturday 21st September, 1957 in the Municipal Travellers Bungalow, Chinglepet.

Dr. P. K. Krishnan Kutty, M.D., M.R.C.P. (Lond) M.R.C.P. (Edin) F.R.F.P. & S. (Glas) addressed on "Problems and diagnosis with reference to cardio Vascular diseases."

COIMBATORE BRANCH

A meeting of the Association was held on Saturday the 20th July, 1957 in the Association premises. Dr. T. V. Sivanandam, M. B. B. S., M. L. C., President, presided.

There was an election for the President and Three Vice-Presidents for the year 1957-58 for the I. M. A. Central.

Dr. A. Ananthanarayana Iyer, B. A., M. B. B. S., M. SC., F. A. SC., Director and Professor of Anotomy, Stanley Medical College, Madras gave a very interesting and informative lecture on "Echoes of Nature's Thythm in Human Life."

* * *

A monthly meeting of the Association was held on Saturday the 17th August, 1957 in the Association premises. Dr. T. V. Sivanandam, M.B.B.S., M.L.C., President presided.

There was an election of Two Vice-Presidents to the Madras State Branch of I. M. A. for the year 1957-58.

Dr. P. Vadamalayan, M.B.B.S., Hony. Surgeon, Government Erskine Hospital, Madurai, gave a lecture on "The Surgery of the Peptic Ulcer,"

MADRAS CITY BRANCH

A meeting was held on Friday the 13th September 1957 at the Madras Medical College.

Dr. A. L. Cochrane, Director, Medical Research Council Pneumoconiosis Research Unit, England, delivered a lecture on "The Epidemiology of some common diseases in Britain."

NILGIRI DISTRICT BRANCH

A monthly meeting of the Association was held at Military Hospital, Wellington, on 24—8—1957.

Lecture on "Medical Problems of Plantations" by Dr. H. C. Berlie, Dr. De Lemos and Dr. P. V. Kurian, was held.

Election of 2 Vice-Presidents to Madras State Branch was held.

RAMNAD BRANCH

A meeting of the Branch was held on 11—8—1957 in Virudhunagar. A condolence resolution was passed on the death of Dr. R. Venkatachary, the President. Dr. Raju Iyer was elected President. Election to Central Office and State Branch was held. Dr. S. Kameswaran, M. B. B. S., D. L. O., spoke on "Recent Advances in E. N. T."

SALEM BRANCH

The monthly meeting of the above Association was held at Foulkes Hall on 27—7—57 with Dr. Rajaram in the chair.

There was an election of the President and the Vice-Presidents for the Central Office.

The house then took up for discussion the matter of furnishing accounts to the Deputy Commercial Tax Officer.

"This association while appreciating the Government for having exempted Medical Practitioners from the purview of Sales Tax according to the G. O. Press No. 1773 Revenue dated 10-7-51 Government Memo No. 9748 N/54-1 dated 8—4—54 from the Board of Revenue Ref. A-3427/54-2 dated 18-5-54 regrets that the members are asked to furnish accounts for '56-'57. This association requests the Government to leave out Medical Practitioners once for all from the purview of the Sales Tax as their outlook is not commercial.

Dr. N. Vaidyanathan spoke on "Fevers - Difficulty in Diagnosis and Management."

SOUTH ARCOT BRANCH

The monthly of the Association was held on Saturday the 24th August 1957 in the Y. M. C. A. Hall, Cuddalore.

Dr. B. Veda, M.B.B.S., Cuddalore spoke on "Diseases of the Chest" and Dr. S. Sankararaman, M.B.B.S., spoke on "Cortisone and A. C. T. H. Therapy.

TRICHY BRANCH

A monthly meeting of the Association was held at 5-30 p.m. on Saturday the 24th August 1957 in the premises of the Medical Association Buildings, Salai Road, Woriur, Tiruchirapalli.

Dr. K. G. Menon, M.B.B.S., presided. Dr. P. A. Menon, FRCS, D.P.H., Southern Railways, Madras and Dr. P. K. Kalyanaraman, M.B.B.S., Hony. Physician, Govt. Hd. Qrs. Hospital, Coimbatore addressed the members on "Joint Stiffness" and "Abdominal Pain" respectively.

Election of 2 Vice-Presidents for the Madras State Branch of I. M. A. for the year 57-58 was held.

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A meeting of the Association was held on the 3rd Sept. 1957 in the Association premises.

Dr. M. S. Narayanan, L.M.S., presided. Dr. S. J. Baker, M.D., Clinical Pathologist, Christian Mission Hospital, Vellore, addressed the members on "Spine Syndrome."